1. How does GABA (gamma-aminobutyric acid) change the neuronal excitability?
2. How does the cell excitability change with the decrease of resting potential?
3. How does the cell excitability change with the increase of resting potential?
4. How the transport of glutamate is changed in ischemic condition in neuronal tissue?
5. How to explain the regeneration of the proximal extremity of the peripheral neurones?
6. The blood-brain barrier prevents pathogens and immune-competent cells entering the nervous system. What toxins can reach neurons in the spinal cord through retrograde axonal transport?
7. What are causes of Waller degeneration?
8. What are causes responsible for cerebral tissue glyosis?
9. What are causes responsible for the damage to oligodendrocyte cells?
10. What are causes that induce the reactivity of ependymal cells?
11. What are consequences of the cross section of the axon?
12. What are consequences of ventricular system inflammation?
13. What are effects of axon transection?
14. What are pathogenetic factors of hypoxic neuronal injuries?
15. What are pathogenetic mechanisms of cerebral edema of vascular origin?
16. What are pathogenetic mechanisms of cerebral tissue glyosis?
17. What are pathogenetic mechanisms of demyelination?
18. What are pathogenetic mechanisms of microglia reactivity to neuronal lesions?
19. What are pathogenetic mechanisms of neurodegenerative diseases?
20. What are pathogenic factors that can trigger neuronal apoptosis?
21. What are pathogenic links in the neuronal demyelination process?
22. What are pathogenic mechanisms of papilledema?
23. What are the causes of cytotoxic edema?
24. What are the causes of vasogenic cerebral edema?
25. What are the components of the Cushing reflex as signs of intracranial pressure increase?
26. What are the effects of prolonged activation of N-methyl-D-aspartate receptor?
27. What are the mechanisms of action of neuroprotective drugs?
28. What are the mechanisms of prolonged activation of N-methyl-D-aspartate receptor?
29. What are the non-specific mechanisms of neuronal degeneration?
30. What are the pathogenetic mechanisms of neuronal apoptosis?
31. What are the pathogenetic mechanisms of neurodegenerative diseases?
32. What are the pathological processes in which excitotoxicity is implicated as pathogenetic mechanism?
33. What are the toxic effects of glutamate?
34. What are variants of dysregulation of nerve impulse transmission in demyelination?
35. What condition leads to reduced cerebral perfusion pressure?
36. What does represent excitotoxicity?
37. What effects induce the distal demyelination of the axon?
38. What factor cause the neuronal hyper-excitability (tetanus syndrome)?
39. What factors can disturb the release of the neurotransmitters into synaptic cleft?
40. What factors cause the neuronal hypo excitability ?
41. What factors decrease the neurotransmitters effects on the postsynaptic neuron?
42. What factors stabilize the neuronal membrane?
43. What intracellular dyshomeostasis is found in neurons during ischemia?
44. What intracellular electrolytic dyshomeostasis is found in the neurons during necrosis?
45. What is pathogenesis of neuromuscular transmission disorder due to local anesthetics?
46. What is pathogenesis of neuronal excitability disorders due to ischemia?
47. What is the basic pathogenetic link of neuronal degeneration?
48. What is the depletion effect of neurotransmitters reserves in nerve endings?
49. What is the depletion effect of noradrenaline reserves into synaptic cleft?
50. What is the effect of norepinephrine reserves depletion in postsynaptic sympathetic nervous ends?
51. What is the impact of cerebral lesions on oligodendrocytes?
52. What is the mechanism of increased cellular excitability in hypoxic conditions?
53. What is the mechanism of increased cellular excitability under hyponutrition state?
54. What is the mechanism of increased excitability of the neuron under the hypoxia?
55. What is the mechanism of neuronal excitability disorder under the conditions of increased lipid peroxidation?
56. What is the specific mechanism of neuronal degeneration?
57. How does the intraneuronal concentration of electrolytes change when switching off of Na+/K+ ATP-ase membrane pumps occurs?
58. How does the neuronal excitability change to increased resting potential?
59. What is the mechanism that explains fixed pupils with lack of reactivity to light in increasing intracranial pressure?
60. What is the mechanism that explains squint due to increased intracranial pressure?
61. What is the microglia reactivity to nerve tissue lesions?
62. What is the pathogenetic chain of neuronal degeneration due to cerebral hypoperfusion?
63. What is the pathogenetic role of intracellular potassium dyshomeostasis in development of neuronal necrosis?
64. What is the pathogenetic role of mitochondrial damage in development of neuronal injury?
65. What is the pathogenetic role of reactive oxygen species (ROS) in development of neuronal necrosis?
66. What is the role of neuroglobin?
67. What is the pathogenicity of neuronal excitability disorders in hypercalcaemia?
68. What is the pathogenicity of neuronal excitability disorders?
69. What is the pathogeny of hypoxic neuronal injury?
70. What is the pathophysiologic mechanism of cytotoxic cerebral edema?
71. What is the pathophysiologic mechanism of vasogenic cerebral edema?
72. What is the role of astrocytes in CNS injury?
73. What is the role of neuroglial cells in CNS homeostasis?
74. What is Waller degeneration?
75. What mechanisms ensure the neuron's survival due to peripheral axons transaction (degeneration)?
76. What process increases the excitability of the neuron?
77. What processes can induce the neuronal degeneration?
78. What processes increase the excitability of the neuron?
79. What stages of neuro-muscular transmission are affected by botulin toxin?
80. What substances disturb the trans-axonal transport of neurotransmitters?
81. Which factors lead to neuronal apoptosis?
82. A distinctive clinical sign for primary hypocorticism is skin hyperpigmentation. What is pathogenesis?
83. A distinctive clinical sign for secondary hypocorticism is skin depigmentation. What is pathogenesis?
84. All three forms of hypocortisolism (primary, secondary and tertiary) represent disorder of hypothalamus - pituitary – adrenal glands axis at different levels. Level disorder r can be determined via hormones dosage into the blood. What is the hormonal pattern in the primary hypocortisolism?
85. At physical examination a patient with hypercorticosolism present with peripheral edema. What is possible mechanism for this?
86. At physical examination a patient with hypercorticosolism presents arterial hypertension. What are possible pathogenetic mechanisms for this?
87. At physical examination of the patient with hypercortisolism revealed: body weight – 90 kg, excessive adipose tissue storages on face (moon face), on trunk, buffalo hump. What is possible pathogeny for these?
88. Biochemical analysis of blood in patients with hypocorticism has showed fasting hypoglycemia - 50 mg dL. What is the pathogenesis?
89. Biochemical analysis of blood in patients with total hypocorticism has showed hyponatremia - 135 mEq / L and hyperkalemia - 5.5 mEq / L .What is the pathogenesis?
90. Biochemical blood analysis in a patient with hypercorticosolism revealed fasting hyperglycemia 10 mmol/L. What are possible pathogenetic mechanisms?
91. Biochemical blood analysis in a patient with hypercorticosolism revealed sodium level 150 mEq/L and potassium level 3,5 mEq/L. What is possible pathogenetic mechanism?
92. Biochemical blood analysis in a patient with hypercorticosolism revealed fasting hyperglycemia 10 mmol/L. What are possible pathogenetic mechanisms?
93. Biochemical hormone profile in a patient with hypercorticosolism reveled: high cortisol level, low ACTH and CRH level. What type of hypercorticosolism is found in the patient?
94. Blood test analysis in a patient with absolute insulin deficiency revealed: erythrocytes – 6,0x1012/L, hematocrit – 60%. What is pathogeny of these changes?
95. Clinical examination of a patient with absolute insulin deficiency revealed dry skin with reduced turgor. In the axillary and inguinal areas there is inflammation with acne, and multiple furuncles. What is pathogeny of furunculosis in hypoinsulinism?
96. Clinical examination of patient N. with hypercorticosolism revealed thin skin with depigmentation and ecchymosis. What is possible pathogeny for these changes?
97. Frequent changes in hypercorticosolism are changes in systemic circulation. What are pathogenetic mechanisms for changes in cardiovascular function in glucocorticoid hypersecretion?
98. Glucocorticoids are involved in control of lipid metabolic processes. What are changes in lipid metabolism in glucocorticoid hypersecretion?
99. Glucocorticoids can modulate inflammatory reaction. What are changes in exudation in inflammatory focus in hypersecretion of glucocorticoids?
100. Glucocorticoids can modulate inflammatory reaction. What are changes in leucocyte migration in inflammatory focus in hypersecretion of glucocorticoids?
101. Glucocorticoids control the protein metabolism. What are changes in protein metabolic processes in glucocorticoid hypersecretion?
102. Glucocorticoids have essential role in carbohydrate metabolism. How carbohydrate metabolic processes are changed in glucocorticoid hypersecretion
103. Glucocorticoids in physiological and pharmaceutical doses influence the immunity. What are changes in specific immunity in glucocorticoid hypersecretion?
104. Glucocorticoids influence the conjunctive tissue. What changes can be attested in the lymphoid tissue in hypersecretion of glucocorticoids?
105. Glucocorticosteroids hormones are instrumental in metabolism of carbohydrates. How does carbohydrate metabolism change in patients with hypocorticism?
106. Glucocorticosteroids hormones have vital importance- their absence leads to death in a few days. What is the vital importance of glucocorticosteroids in extreme conditions?
107. Glucose tolerance is the ability of the body to assimilate glucose and maintain blood sugar level. What is pathogeny for reduced glucose tolerance in insulin deficiency?
108. Glucose up-take from the blood depends on membranary transporters (GLUT 1-4) which can be insulindependent or insulin-independent. What cells are equipped with insulin-dependent transporters?
109. Glucostatic mechanisms which control glycemia are the hyperglycemiant and hypoglycemiant effects. What is compensatory hypoglycemiant factor which can be involved in hyperglycemia?
110. Glucostatic mechanisms which control glycemia are the hyperglycemiant and hypoglycemiant effects. What are compensatory hyperglycemiant factors which can be involved in hypoglycemia?
111. Hematologic examination in a patient with diabetes mellitus type I revealed presence of 12% of glycolizated hemoglobin. What is the clinical significance for presence in the blood of glycolizated hemoglobin?
112. How do glucocorticoids act on vascular reactions during inflammation?
113. How do glucocorticosteroid hormones influence the process of leukocyte migration in the inflammatory reaction?
114. How does acid-base balance change in patients with type I diabetes mellitus?
115. How does allergic reactivity modify in patients with hypocorticism?
116. How does blood biochemistry change in GH hypersecretion in children?
117. How does body weight change in diabetes mellitus type I?
118. How does carbohydrate metabolism change in GH hypersecretion in children?
119. How does lipid metabolism change in hypothyroidism?
120. How does lipidogramm change in patients with type I diabetes mellitus?
121. How does metabolism change in diabetes mellitus type I?
122. How does protein metabolism change in GH hypersecretion in children?
123. How does protein metabolism change in patients with type I diabetes mellitus?
124. How does the skeletal muscles weight change in diabetes mellitus type I?
125. How does urine output change in diabetes mellitus type I?
126. How is hyperaldosteronism manifested?
127. How is hypoaldosteronism manifested?
128. How metabolic processes are changed in insulin deficiency
129. How the carbohydrate metabolism is modified in somatotropin hypersecretion?
130. How the secondary hyperthyroidism is manifested clinically?
131. How the tertiary hyperthyroidism is manifested clinically?
132. Hyperaldosteronism has more pathogenetic mechanisms on which depend therapeutic approach. What is pathogeny of hyperaldosteronism in renal ischemia?
133. Hyperaldosteronism has more pathogenetic mechanisms on which depend therapeutic approach. What is pathogeny of hyperaldosteronism in hypovolemia?
134. Hyperaldosteronism has more pathogenetic mechanisms on which depend therapeutical approach. What is pathogeny of hyperaldosteronism in renin-secreting tumor?
135. Hyperaldosteronism has more pathogenetic mechanisms on which depend therapeutical approach. What is pathogeny of hyperaldosteronism in liver failure?
136. Hypercorticosolism is associated with mineral dyshomeostasis. How calcium metabolism and bones are affected in glucocorticoid hypersecretion?
137. Hyperglycemia is a specific symptom for absolute insulin deficiency and is the result of two processes: intense mobilization of glucose from storages and reduced uptake of glucose in peripheral tissues. What is pathogeny for reduced peripheral up-take of glucose in peripheral tissues?
138. Hyperglycemia is a specific symptom for absolute insulin deficiency and is the result of two processes: intense mobilization of glucose from storages and reduced uptake of glucose in peripheral tissues. What is the pathogeny of intense glycogenolysis in insulin deficiency?
139. Hyperglycemia is the main symptom in absolute insulin deficiency. What is its pathogeny?
140. Hyperglycemia is the main symptom in insulin deficiency. What are immediate consequences in hyperglycemia?
141. Hypersecretion of glucocorticoids affects electrolytic homeostasis. What are pathogenetic mechanisms for electrolytic dyshomeostasis in glucocorticoid hypersecretion?
142. In C patient suffering from primary hypocortisolism objective was observed hyperpigmentation of skin. What is the pathogenesis?
143. In diabetes mellitus in lack of insulin there is catecholamine hypersecretion. What are the metabolic effects of high catecholamine level? 97
144. In diabetes mellitus in lack of insulin there is glucagon hypersecretion. What are the metabolic effects of high glucagon level?
145. In diabetes mellitus in lack of insulin there is glucocorticoid hypersecretion. What are the metabolic effects of high glucocorticoid level?
146. In diabetes mellitus in lack of insulin there is glucocorticoid hypersecretion. What are the metabolic effects of high glucocorticoid level?
147. In glucocorticoid hypersecretion there develop digestive disorders. What are changes in digestive function in hypersecretion of glucocorticoids?
148. In hypercorticosolism there is specific adipose tissue deposition – “moon face”, “buffalo hump”, central obesity. What is the pathogeny for enhanced lipogenesis in these regions? 106
149. In insulin deficiency there is intense mobilization of lipids from storages. What are consequences of transport hyperlipidemia?
150. In insulin deficiency there is intensification of catabolic processes. What hormones are responsible for intensification of catabolism in absolute insulin deficiency?
151. In patient D was established primary hypercorticosolism. Radiography revealed adenoma in the left adrenal gland and atrophy of right adrenal gland. In patient was performed left adrenal adenoma resection. What postsurgical complication is possible in this situation?
152. In patient D was established primary hypercorticosolism. Radiography revealed adenoma in the left adrenal gland and atrophy of right adrenal gland. What can be the life-threatening consequences after adenoma resection?
153. In patients with diabetes mellitus there is necessary to control lipid homeostasis, as lipid metabolic disorders can lead to late complications in these patients. What are changes in lipid spectrum in the blood in absolute insulin deficiency?
154. Incorrect treatment of diabetes mellitus type I which can’t maintain glycemia, complicates with albuminuria. What are pathophysiological mechanisms of albuminuria in absolute insulin deficiency?
155. Insulin changes glycemia and peripheral use of glucose. What are the mechanisms for high peripheral up-take of glucose induced by insulin?
156. Insulin deficiency and hypersecretion of glucagon in diabetes mellitus lead to transport hyperlipidemia and liver invasion with free fatty acids. What can be the consequences?
157. Ketonemia is a characteristic sigh in absolute insulin deficiency. What can be the consequences of excessive accumulation of ketone bodies?
158. Laboratory tests in a patient with absolute insulin deficiency revealed hyperlipidemia with high level of VLDL and LDL. What is pathogeny for these changes?
159. Laboratory tests in a patient with absolute insulin deficiency revealed hyperlipidemia with high level of non-esterified free fatty acids. What is pathogeny of these changes?
160. Lack of glucocorticoid hormones clinically is manifested by vascular disorders. How is maintained the normal muscle tone in patients with hypocorticism?
161. Lack of glucocorticoid hormones clinically is manifested by vascular disorders. How does vascular tone change in glucocorticosteroids hyposecretion?
162. Lack of glucocorticoid hormones is manifested clinically by disturbances of heart function. How do cardiac functions disturb in hypocorticism?
163. Lack of insulin is the main pathogenic loop in diabetes mellitus. How is affected the function of other endocrine gland in insulin deficiency?
164. Leucocytes are affected in diabetes mellitus. What are the consequences for leucocytes in absolute insulin deficiency?
165. Metabolic disorders in absolute insulin deficiency depend on adipose cells as well. What metabolic disorders are disturbed in the adipocytes in insulin deficiency?
166. Metabolic disorders in absolute insulin deficiency depend on adipose cells as well. Why in lack of insulin adipose cells can’t perform glucose metabolism?
167. Metabolic effects of glucocorticoids lead to metabolic dyshomeostasis. What are characteristic biochemical changes in the blood in glucocorticoid hypersecretion?
168. Monitoring of diabetes mellitus involves control of glucosuria. What is pathogeny of glucosuria in lack of insulin?
169. One hypoglycemiant factor which is involved in hyperglycemia is insulin. What are hypoglycemiant mechanisms of insulin?
170. One of compensatory hyperglycemiant factor with high role in hypoglycemia is glucagon. What is the hyperglycemiant mechanism of glucagon?
171. One of medical emergency in people with diabetes mellitus is pH disbalance. What characteristic pH disorders develop in absolute insulin deficiency?
172. One of the vital risks of the stress in people with hypocorticism are hemocirculatory complications - arterial collapse, acute circulatory insufficiency. What is pathogenesis? How can prevent this complication in patients with hipocorticism?
173. One remarkable effect of glucocorticoids is hyperglycemia. What is pathogeny of hyperglycemia in glucocorticoid hypersecretion?
174. One severe medical emergency in absolute insulin deficiency is ketoacidotic coma. What is pathogeny?
175. Patient C., 32 years with a presumable diagnostics of hyperthyroidism . In the blood antibodies against TSH receptors are found. What are the principles of pathogenetical therapy of autoimmune hyperthyroidism?
176. Patient C., 32 years with hyperthyroidism. Biochemically: hyperlipidemia with VLDL and LDL. What are consequences of hyperlipidemia in hyperthyroidism?
177. Patient C., 32 years with hyperthyroidism. Complains muscular weakness and fatigue. Test of muscular force - negative: no standing from chair without hands help. Objectively –leg muscle atrophy. What is pathogenesis of these symptoms?
178. Patient C., 32 years with hyperthyroidism. Complains sensation of worm and intolerance to high temperature. Objectively: body temperature –37,3°C. What is the plausible pathogenesis of hyperthermia?
179. Patient C., 32 years with hyperthyroidism. Complains: body weight loss (5 kg/3 months) although appetite is increased. What is the plausible pathogenesis of hyperthyroidism induced appetite stimulation?
180. Patient C., 32 years with hyperthyroidism. Complains: body weight loss (5 kg/3 months) although appetite is increased. What is the plausible pathogenesis of hyperthyroidism induced body weight loss?
181. Patient C., 32 years with hyperthyroidism. Functional tests indicate an increase of oxygen use and basal metabolism. What are the consequences of the excessive oxygen use?
182. Patient C., 32 years with hyperthyroidism. What supplementary investigations are needed for differentiation of the thyroid hypersecretion patterns: tertiary, secondary or primary?
183. Patient C., 24 years old, presented to the endocrinologist with the presumptive diagnosis “diabetes mellitus type I”. What are the signs that differentiate the diabetes type I from type II?
184. Patient C., 24 years old, presented to the endocrinologist with the presumptive diagnosis “diabetes mellitus type I”. What investigations are necessary to confirm the diagnosis
185. Patient C., 24 years old, presented to the endocrinologist with the presumptive diagnosis “diabetes mellitus type I”. What are the distinctive signs between insulin-deficiency and insulin-resistance?
186. Patient D, known with hypercorticosolism, complains about muscular weakness and fatigability. What are possible pathogenetic mechanisms?
187. Patient D, known with hypercorticosolism, suffered a fracture of lumbar vertebra. What are the possible pathogenetic mechanisms?
188. Patient D. with diabetes mellitus, complains about visual disturbances: reduced visual acuity, blurred vision. What is pathogeny?
189. Patient D., with atrophy of pituitary gland after hemorrhagic shock develops symptoms of hypothyroidism. What processes of thyroid gland were abolished due to lack of TSH?
190. Patient suffering from secondary hypocortisolism complaints weight loss- for 1 year lost 25 kg body weight. Objective - thinning of subcutaneous fat. What is the pathogenesis of fat atrophy in hypocortisolism?
191. Patient suffering from total hypocortisolism complaints muscle weakness, fatigue. What is the pathogenesis?
192. Patient T, with Cushing syndrome, complains about lack of children. What is possible pathogenetic mechanism?
193. Patient T. with diabetes mellitus complains about retrosternal pain with characteristic irradiation for coronary insufficiency. What is pathogeny?
194. Patients suffering from hypocorticism complaints vertigo when getting up from bed sometimes syncope. BP - 80/40 mm Hg, pulse - 120 beats / min. What is the possible pathogenesis of tachycardia in hypocorticism?
195. Patients suffering from hypocortisolism complaints vertigo when getting up from bed, sometimes syncope. BP - 80/40 mm Hg, pulse - 120 beats / min. What is the possible pathogenesis of decreased diastolic blood pressure in hypocortisolism?
196. Patients suffering from secondary hypocortisolism complaint muscle weakness, fatigue. Objective atrophy is found in skeletal muscle. What is the pathogenesis?
197. Primary and secondary hypocortisolism have most analog clinical manifestations. Which is a characteristic clinical manifestations for primary hypocortisolism?
198. The biochemical findings of the 24 years old patient C., with diabetes mellitus type I revealed hypercholesterolemia. What is the pathogenesis?
199. The biochemical findings of urine at 24 years old patient C., with diabetes mellitus type I revealed the presence of albumin concentration of 200 mg / L. What is the pathogenesis?
200. The biochemical findings of urine at 24 years old patient C., with diabetes mellitus type I revealed the presence of glucose. What is the pathogenesis?
201. The implication of glucocorticosteroids in carbohydrate metabolism tangentially reach and other endocrine glands. How does insulin and glucagon secretion change in hypocorticism?
202. The patients, 23 years old, that suffers chronic non-specific a long time was treated with high doses of glucocorticosteroids, subsequently radiography has detected both adrenal glands atrophy. What is pathogenesis?
203. There is clinical significance in differentiation of the type of hyperaldosteronism. What can be the causes for secondary hyperaldosteronism?
204. There is clinical significance in differentiation of the type of hyperaldosteronism. What can be the cause for primary hyperaldosteronism?
205. There is known that neurons are not affected in diabetes mellitus. What conditions are responsible for glucose up-take by neurons in lack of insulin?
206. What are biochemical changes in the blood in insulin deficiency?
207. What are causes of polydipsia in diabetes mellitus type I?
208. What are characteristics of inflammatory reaction in hypersecretion of glucocorticoids?
209. What are circulatory changes in hyperaldosteronism?
210. What are manifestations of ADH hypersecretion?
211. What are manifestations prolactin hypersecretion in males/females?
212. What are pathogenetic mechanisms for polydipsia in absolute insulin deficiency?
213. What are pathogenetic mechanisms for reduced microbicidal activity of phagocytic cells in insulin deficiency?
214. What are the causes of ADH hypersecretion?
215. What are the causes of functional disorders of the adenohypophysis?
216. What are the causes of growth hormone hypersecretion?
217. What are the causes of growth hormone hyposecretion?
218. What are the causes of impaired function of anterior pituitary?
219. What are the causes of prolactin hypersecretion?
220. What are the changes in the blood in case of hypercortisolism?
221. What are the consequences of glycolization of collagen in the vascular basement membrane?
222. What are the consequences of inactivation of GLUT4 insulin-dependent receptors on skeletal muscles in absolute insulin deficiency?
223. What are the effects of VLDL and LDL glycolization in long-term hyperglycemia?
224. What are the extrapituitary causes of adenohypophysis functional disorders?
225. What are the factors which contribute to development of clinical manifestations in tertiary hypercorticosolism?
226. What are the factors which contribute to development of clinical manifestations in primary hypercorticosolism?
227. What are the factors which contribute to development of clinical manifestations in secondary hypercorticosolism?
228. What are the manifestations of glucocorticoid hypersecretion/hyposecretion?
229. What are the manifestations of primary hyposecretion of glucocorticosteroids?
230. What are the manifestations of tertiary hyposecretion of glucocorticosteroids?
231. What are the pathogenetic principles in therapy of tertiary hypercorticosolism?
232. What are the principles of direct regulation (downward) of axis hypothalamus - pituitary – adrenal glands?
233. What conditions enhance glucose use by neurons in the lack of insulin?
234. What hematological changes can be found in hypercorticosolism/hypocortisolism?
235. What is a cause of decreased bactericide activity of phagocytes reduction in patients with type I diabetes mellitus?
236. What is a cause of hypersthenuria in diabetes mellitus type I?
237. What is a cause of muscle atrophy in type I diabetes mellitus?
238. What is a cause of slow wound regeneration in patients with type I diabetes mellitus?
239. What is a mechanism of albuminuria in patients with type I diabetes mellitus?
240. What is pathogenesis of hyperglycemia in diabetes mellitus type I?
241. What is pathogenesis of negative nitrogen balance in type I diabetes mellitus?
242. What is pathogenesis of polyuria in diabetes mellitus type I?
243. What is pathogenetic mechanism for delayed wound healing in patients with diabetes mellitus?
244. What is pathogenetic mechanism for hypersthenuria in absolute insulin deficiency?
245. What is pathogenetic mechanism for polydipsia in absolute insulin deficiency?
246. What is pathogenetic mechanism for polyuria in absolute insulin deficiency?
247. What is the cause of antidiuretic hormone hypersecretion/hyposecretion?
248. What is the cause of GH hypersecretion?
249. What is the cause of GH-releasing hormone hyposecretion?
250. What is the cause of impaired function of anterior pituitary?
251. What is the cause of prolactin hypersecretion?
252. What is the manifestation of glucocorticoid hyposecretion?
253. What is the mechanism of ADH hyposecretion in trauma of pituitary pediculus?
254. What is the mechanism of adrenal hyposecretion in a prolonged administration of high doses of glucocorticosteroids?
255. What is the mechanism of glucosuria in patients with type I diabetes mellitus?
256. What is the mechanism of prolactin hypersecretion in the trauma of the pituitary peduncle?
257. What is the pathogenesis of primary hypothyroidism in autoimmune thyroiditis with antibodies against TPO (thyroperoxidase I)?
258. What is the possible cause of primary hypercortisolism?
259. What is the possible cause of secondary hypercortisolism?
260. What is the role of glucocorticoid hormones in the development of inflammatory reaction?
261. What is the somatic manifestation of GH hyposecretion in adults?
262. What is the somatic manifestation of GH hyposecretion in children?
263. What liver metabolic functions are affected in absolute insulin deficiency?
264. Where is known that patients with insulin deficiency have tendency to develop pyogenic infections. What is pathogeny?
265. Which of the following are cardiac disorders in hyperthyroidism ?
266. Which of the following are cardiac disorders in hypothyroidism ?